10.—Poison plants in Western Australia and colonizer problem solving

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Abstract

Because of their leguminous seed pods, poisonous plants of the genera *Gastrolobium* and *Oxylobium* posed major problems for Western Australia's nascent pastoral industry. Not only did they cause considerable economic loss, but it took nearly twelve years for their lethal properties to be recognised. This delay is attributed here to the nature of these plants and to the confusion generated among colonists by the conflicting explanations offered by Dr Joseph Harris and the botanist James Drummond.

The process by which the toxic nature of these plants was established raises important theoretical implications about problem solving and hazard response in unfamiliar environments and throws light on colonizer adjustment. It is concluded that learning can be viewed as a progression along a generalization-differentiation continuum where generalization denotes the constraining effect of well established past behaviours and differentiation refers to a growing sensitivity and responsiveness to previously unfamiliar environmental stimuli.

Introduction

Any relocation in space will induce a period of active adjustment. Nowhere is this more evident, or more crucial than in pioneering situations. Here, people with diverse backgrounds, predispositions and expectations invariably encounter grossly unfamiliar and often extremely forbidding conditions which contain few of the elements which gave their former behaviour structure and cohesion. Yet, with the exception of Found's exploratory theorizing of incremental learning (Found 1971, p.139-141), colonizer adjustment has received little attention.

No attempt is made here to examine all types of learning evident in colonizer adjustment. Rather, the emphasis is placed on the problem solving activities associated with Western Australia's poison plants for these activities represented a deliberate attempt to decrease the disparity between the expected and actual outcomes of pastoral operations. This is a unique example but does have important theoretical implications. The paper is therefore structured into three parts which focus respectively on the nature of the problem, the sequence of events leading to its resolution, and the characteristics of problem solving behaviour evident in this sequence.

The problem in its context

Western Australia has more than 150 endemic plants capable of poisoning stock under some circumstances (Gardner and Bennetts 1956). Of these, the 32 toxic species of the genera *Oxylobium* and *Gastrolobium* are the most widespread and lethal, less than 15 g being at times sufficient to kill an adult sheep (Aplin 1967). As partly indicated in Table 1, they constituted the major hazard for the nascent pastoral industry, losses from them exceeding combined losses from all other hazards including bushfires, floods, drought, aboriginal depredations and the attacks of native dogs. Stock losses were recorded as early as December 1830 and continued at a high level throughout the 1830s. They created the conditions of stress necessary for inducing accelerated learning and active problem solving (Festinger 1964; Heider 1958; Lewin 1938) but the cause of death was not positively confirmed until May 1841 (*Inquirer*, 26 May 1841; *Perth Gazette* (hereafter P.G.), 17 May 1841). Two factors, the nature of the problem, and the nature of the problem solvers, account for this slow resolution.

The nature of the problem

A recognition of the fact that the genera *Oxylobium* and *Gastrolobium* are members of the pea flowered family (Papilionaceae) is fundamental to understanding delays in identification. Shepherds believed their leguminous structure placed them among the more nutritious local fodder sources and deliberately sought them out. As the plants had not been previously encountered in Australia, there could be no warning of their lethal properties. To add further confusion, only 15 of the 59 species recognised by 1864 (Bentham 1864, p. 14-26, 96-207) have since been found to be toxic (Gardner and Bennetts 1956, p. 52-75). All species can be eaten, their palatability being greatest in the winter and spring months when toxicity is at a peak. These features alone sufficiently explain delays but they were exacerbated by the nature and effect of the toxic element.

The toxic agent of *Oxylobium* and *Gastrolobium* is monofluoroacetic acid, found elsewhere only in the gifblaar (*Dichapetalum cymosum*) of South Africa and the gigyea (*Acacia georgina*) of Queensland and the Northern Territory (Aplin 1967). More commonly known as the rabbit poison '1080' (its sodium salt derivative), fluoroacetic acid is odourless, colourless, tasteless, water soluble and extremely stable. When ingested, it converts by enzyme action into the toxic fluorocitric acid (Peters 1954). As shown in Table 2, only small dosages are required to produce fatal results. There is no known antidote. As its presence in nature was not demonstrated until 1943, settlers could not know of its existence.

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Table 1

Recorded stock losses from poison plants, 1833-1840

Date recorded	Details of mortality	Location	Remarks
(PG) 21 September 1833	Heavy stock losses: upwards of 100 sheep from 1 flock.	Upper Swan	
(PG) 19 October 1833	4 out of 6 bullocks. Dog eating one of the bullocks later died.	York Road	
(PG) 15 March 1834	21 sheep	Upper Swan—Scarp face	
(SD) 17 March 1834	300+ sheep, 5 cattle, 3 horses	King George Sound	
(PG) 16 August 1834	40 sheep, 6 goats	Upper Swan	
(PG) 11 April 1835	55 out of 66 goats	York Road	Very dry season with the result that many flocks were moved to the inland pastures of the Avon Valley. Total sheep numbers were then less than 4 000.
(PG) 16 May 1835	93 sheep, 13 goats, 6 bullocks	York Road	
(PG) 20 June 1835	15 sheep	York Road	
(PG) 14 November 1835	8 out of 10 bullocks	York	
(PG) 26 November 1836	130 sheep out of 300	Williams district	
(PG) 4 August 1838	56 sheep out of 239	Williams district	Exceptionally dry season which persisted until the winter of 1842
(PG) 20 November 1838	Major losses of cattle	Williams district	
(PG) 7 July 1839	Major losses of all stock throughout the colony.		
(PG) 28 March 1840	250 sheep and 12 cattle out of 750 sheep and 54 cattle.	Kojonup district	Mortality associated with the overland movement of imported stock from King George Sound to the Avon Valley.
(PG) 27 June 1840	63 sheep out of 600. Other losses were even greater.	Road from King George Sound to the Avon Valley	
(PG) 10 October 1840	33 out of 180 sheep	Upper Swan—Scarp face	
(INQ) 7 October 1840	Total flock of 180 sheep	York	
(PG) 5 December 1840	All of Craigie's flock (c.500) 304 of Tapsou's flock 118 of MacDonald's flock	Kojonup district	

Source: *Perth Gazette* (shown as PG); *Inquirer* (INQ); *Spencer Diary* (SD).

Table 2

Toxicity of sodium fluoroacetate (50% *mortality*)

Animal	Dosage (a) (mg per kg of body weight)
Dog	0.066
Sheep	0.25
Pig	1.0
Horse	1.0
Kangaroo	8.0 (b)
Pigeon	9.0
Frog	300.0

(a) All dosages were administered orally.

(b) This figure is for 100 per cent mortality. Dosage for 50 per cent mortality is not available.

Source: Department of Agriculture, Western Australia, n.d., *Sodium fluoroacetate: A resume of the available literature dealing with the poisoning of human beings and animals* (mimeod).

Most English poisonous plants, by contrast, are cyanogenetic, or contain poisonous, sometimes narcotic, alkaloids. With the exception of foxglove (*Digitalis purpurea*), their poisonous qualities were suggested through their acrid taste and the foetid smell of their sap and bruised leaves. These characteristics settlers were well aware of and it is not surprising that attention first centred on a number of cyanogenetic plants, particularly the blind grasses (*Stypandra imbricata* and *S. grandiflora*) which induce a range of symptoms similar to those induced by *Oxylobium* and *Gastrolobium* (Gardner and Bennetts 1956, p. 79-82), and the narcotic Woodbridge poison (*Isotoma hypocrateriformis*), a member of the lobelia family.

The rarity of fluoroacetic acid naturally is matched by its variable toxicity, variations depending on the species, its location, and the time of the year (Aplin 1967). These variations, in turn, induce a wide range of symptoms from mild agitation, partial paralysis and blindness, to a violent, convulsive death (Carne, Gardner and Bennetts 1926). This feature most perplexed colonists because several conditions closely corresponded to already known stock diseases, particularly 'hoove', 'staggers', and 'blood striking', all of which were caused by an inability to digest over-rich herbage (see Wilson 1852, v. 2, p. 684-6). In addition, not all animals were equally affected (Table 2). Sheep and goats appeared most susceptible, followed by cattle. Horses had a high resistance (Landor 1847, p. 379-380) which was enhanced by their greater body weight. The relative immunity of local fauna was most perplexing. The botanist James Drummond consistently refused to accept that York Road poison (*G. calycinum*) had poisonous properties for he had observed pigeons feed on the seeds of the plant with immunity (P.G., 5 December 1840).

Problem solvers

While pastoralists were extremely anxious to identify the cause of mortality, and while they were deeply involved in the poison debate, they generally deferred to the superior knowledge and expertise of Joseph Harris, a surgeon with veterinary experience, and James Drummond. Similarly, the Agricultural Society took little direct action although it did provide a forum for discussion and debate. As a consequence, although their initial expertise was only marginally superior, Drummond and Harris emerged as the major authorities and the key problem solvers. That they held strongly divergent views greatly added to the confusion surrounding stock deaths.

Harris was convinced that stock died from 'blood striking' (P.G., 21 September 1833). His conclusions were derived from detailed patho-

logical evidence and his knowledge of English stock diseases but there were other considerations. Apart from being Drummond's rival for prestige and influence, Harris was concerned that the colony's already bad reputation would be further degraded by reports of high stock fatalities. This attitude, first expressed in September 1833 (P.G., 2 September 1833), was maintained throughout the 1830s with the result that unfavourable evidence was unconsciously suppressed.

Drummond, by contrast, early suspected plants because of the similarity of the vegetation associations in localities where deaths were reported (P.G., 19 October 1833; 5 December 1833). Drummond's approach involved the isolation, identification, description and testing of suspected species (P.G. 3 February 1838) but was slow to produce results for he began by isolating plants similar to those known to be toxic in England. So convinced was Drummond of the beneficial nature of all legumes that the pea-flowered shrubs of *Gastrolobium* and *Oxylobium* were only examined after all other alternatives had been examined and rejected (P.G., 24 August 1838; 5 December 1840).

Learning sequence

The discovery of poison plants was largely a result of trial-and-error procedures. Although existing knowledge and conceptual structures pointed to several lines of enquiry, the conflicting explanations offered by Drummond and Harris made the initial task one of definition. Extinction of established learning was a necessary concomitant. Until the problem had been defined, there could be no effective attempts at its solution. Learning was not characterized by steady increments. Rather, it progressed in spurts, these corresponding with 'crisis states' or periods of considerable stress. Consequently, the record of stock losses shown in Table 1, even though these are incomplete, identify the major periods. Greatest concern and most active problem solving were associated with those areas and time periods where mortalities posed serious threats. That there should be periods of relative quiescence indicates that settlers in established areas had learned to live with the threat of poison and avoid it, and suggests that improvised techniques, determined by trial-and-error, compensated for detailed knowledge and understanding.

Attempts at definition

Major concern was first expressed in the winter of 1833. Two factors induced this. Firstly, sheep which had previously been pastured on the alluvial flats along the Swan and Canning Rivers were now being grazed on the edge of the Darling Scarp (Moore 1884, p. 210). Here, they came in contact with Champion Bay poison *(G. oxylobioides)* and prickly poison *(G. spinosum)* and fatalities increased rapidly. This was sufficient cause for alarm as it is unlikely that total sheep numbers then exceeded 3 000, but these fatalities seriously interfered with the intentions of a number of settlers to increase their flock size to the point where they could profitably begin wool production (P.G., 18 May 1833). Harris was requested to outline the symptoms of and treatment for 'blood striking' at the September meeting of the Agricultural Society (P.G., 21 September 1833, 28 September 1833).

Harris' statement failed to dispel fears for within a month Bland, the government stockkeeper, requested Drummond to examine the vegetation in an area where a number of separate fatalities had been recorded. Drummond found nothing of significance but suspected a mineral spring (P.G., 19 October 1833). Harris, who was also consulted, was adamant that the richness of the vegetation was responsible. In this he was supported by a number of settlers who were most perturbed that plants were already being thought to be poisonous (P.G. 26 October 1833).

Further debate was initiated in the following March when Brockman lost 21 sheep (P.G., 15 March 1834). Although he originally consulted Harris (P.G., 21 September 1833), he now seriously doubted that 'blood striking' was the cause because more sheep were affected than could be expected and dogs died after eating the meat of infected carcases. Most importantly, deaths occurred when grass was dry and scarce whereas, in England, they occurred when pastures were abundant and green (P.G., 22 March 1834). His argument was given added force by an anonymous correspondent in the *Perth Gazette* who pointed out that the presumably rich herbage was "long, stringy, spiry grass, suddenly drawn up by the warm forcing sun, and thus brought forward unnaturally" (P.G., 2 August 1834).

Now under strong attack, Harris suggested that the Agricultural Society contact the sister society in New South Wales for its opinion of the disease and its recommended treatment (P.G. 10 May 1834). The reply, published in the *Perth Gazette* on 16 August, was of no value for the full circumstances of the disease were completely unknown outside Western Australia. In his accompanying commentary, Harris admitted some uncertainty about the cause but insisted that the antidotes he proposed, namely bleeding and saline purging, were quite adequate. Therefore there was no cause for alarm (P.G., 16 August 1834).

His optimism seemed justified as fatalities again subsided. They flared up again with increased severity early in 1835 following the general movement of sheep from the coast to the inland pastures of the Avon Valley surrounding York, 80 km to the east (P.G., 8 November 1834, 6 December 1834, 10 January 1835). This had been precipitated by the excessively dry winter of 1834 (P.G., 16 May 1835; Moore 1884, p. 243-248) but, until the road from Guildford to York was completed in October 1835 (P.G., 10 October 1835), stock had to traverse the lateritic-capped hills, broad, sandy slopes and swampy lowlands of the western plateau, over the greater part of which grew luxuriant stands of Champion Bay, York Road and prickly poisons. Stock losses were general (P.G. 4 Novem-

ber 1835). It may have been rough justice but the most severe losses were experienced by Harris. On one crossing, he lost 93 sheep, all 15 goats and 2 bullocks. Nevertheless, he maintained his original position (P.G., 16 May 1835).

Avoidance

The combined and disastrous impact of this new outbreak led to the formulation of effective techniques of avoidance. By June 1835, it was suggested that losses could be substantially reduced by "a careful attention to folding at night, and a fast driving by day, except on the burnt ground, where the herbage is generally young and sweet" (P.G., 20 June 1835). Techniques were further evolved by November when Trimmer advocated a strong application of stock salt prior to departure and muzzling while enroute (P.G. 14 November 1835). By the following June, Bland argued that most effective preventative was the hand feeding of stock for two days prior to departure and then throughout the journey (P.G., 4 June 1836). With the adoption of these practices, fatalities again subsided, the trend being hastened with the clearing of established camp sites along the completed York Road. Once in the Avon Valley, stock were relatively secure because the poisonous plants were restricted to the rugged and rocky hill tops and the sand plain well to the east (Aplin 1967).

Resolution

The excessively dry conditions beginning at the end of 1837 precipitated further outbreaks in widely separated localities as sheep and cattle spread in search of pasture. The considerable anxiety these generated increased the possibility that an effective solution would be found as suspicion was now strongly centred on the poisonous rather than the nutritive qualities of the vegetation. Even Harris was forced to conclude that blind grass may have been the cause of death of sheep he moved to his son's grant on the Williams River in November 1836 (P.G., 26 November 1836). Earlier, Bland had concluded that one of the Gastrolobiums was responsible for he had seen sheep feed on these before going blind or dying (P.G., 4 June 1836). Whitfield had come to a similar conclusion (Erickson 1969, p.54). However after careful examination, Drummond stated that none was harmful. Woodbridge poison was the cause of death (P.G., 3 February 1839). His evidence, presented in the *Perth Gazette* on 10 February 1839, seemed irrefutable. Woodbridge poison was a member of the lobelia family (Lobeliaceae) and this was widely known to contain noxious attributes. He had noticed that no wild animals ate it but an examination of the area in which sheep and goats recently died revealed that a considerable amount had been consumed. The results of a post-mortem examination seemed conclusive: the contents of the stomach smelt very like the plant and had the same acrid odour so common in Engish poisons.

Drummond's findings did not allay fears for losses continued at a disastrously high level. The autumn of 1839 was the driest yet experienced and was succeeded by an abnormally dry winter (P.G., 7 July 1839, 5 October 1839). The drought seriously interfered with attempts to establish an overland stock route from King George Sound to the Avon Valley, and, on one trip, Eyre lost over 250 sheep and 54 bullocks (P.G., 28 March 1840). An acrimonius debate ensured between Drummond, Harris and their supporters, all of whom reverted to their original positions (P.G., 24 March 1839, 28 April 1839, 5 October 1839, 11 April 1840) and this notwithstanding the fact that Harris lost 63 sheep on his trip north from King George Sound (P.G., 27 June 1840).

Matters were brought to a head by the rash of deaths in the newly opened Kojonup District. Here, during September and October 1840, three flocks numbering more than 900 sheep were almost totally destroyed (P.G., 5 December 1840; *Symers Papers*, 5 September 1840). Drummond was consulted and administered a potion from Woodbridge poison. This gave negative results (*Inquirer*, 17 March 1841). Influenced by one of the shepherds, he reluctantly tried York Road poison but, having failed to prove his initial hypothesis, departed for Williams before the results were known. The sheep died the following afternoon (*Government Gazette*, 25 December 1840; P.G., 5 December 1840, 26 December 1840).

When he arrived at Williams, Drummond was contacted by Harris' son who was extremely perturbed by the continuing fatalities in his flock. Close examination of the area where the sheep had been feeding revealed the presence of York Road poison. The evidence now seemed overwhelming for, not only was this poison common throughout the Kojonup District, but Drummond had earlier observed it alongside the York Road where fatalities had been particularly heavy. Tender young branches were collected, made into a drench and given to a healthy goat. It died within 14 hours (P.G., 21 November 1840, 5 December 1840).

The matter was not yet resolved, however. Harris reluctantly accepted his son's and Drummond's evidence but refused to accept that more than one species was involved or that York Road poison was widely distributed, (P.G., 19 December 1840). His view was influenced by a desire to defend the pastoral properties of the Kojonup District. In this he had several supporters including the German botanist Preiss, recently arrived in the colony, who insisted that "leguminous plants are particularly suited for the food of animals and the human race". To prove his point, he drank a wineglass of diluted fluid extracted from the leaves of York Road poison (*Inquirer*, 17 March 1841). Suffering no ill-effects, he recommended the plant to stock holders as "the very best thing they could cultivate as artificial food for stock" (quoted in Erickson 1969, p.59).

To resolve all doubts and end the confusion, the Agricultural Society requested Drummond in May 1841 to demonstrate the toxic properties of the plants he had identified. All three test animals died within four hours. Three dogs also died after feeding on the entrails of the poisoned sheep. Harris was now fully convinced

and agreed that "the plant is a most powerful poison". He knew no antidote (*Inquirer*, 19 May 1841; P.G., 17 May 1841).

Extended learning

Further experiments on 13 August 1841 confirmed these findings and led to the search for an antidote (P.G., 14 August 1841). Within a week it was reported that washing soda may be beneficial (P.G., 21 August 1841). A considerable hiatus followed which suggests that identification of the cause substantially reduced settlers' concern (see P.G., 22 January 1842). A repetition of the May 1841 experiment twelve months later produced negative results and led Drummond to conclude that toxicity was greatest from the beginning of new growth until the onset of the summer drought. January through to May could be considered the quiescent period (*Inquirer*, 18 May 1842). The range of poison plants was now extended to include rock poison (*G. callistachys*) and box poison (*O. parviflorum*) (P.G., 14 May 1842). Heart-leaved poison (*G. bilobum*) growing in the south and particularly around King George Sound was already under suspicion (*Inquirer*, 23 December 1840; P.G., 5 December 1840).

With Drummond's positive identification, fatalities rapidly diminished and it became common practice for shepherds to carry branches of known or suspected species to aid in their identification. Where belts of poison had to be traversed, stock were driven in haste and were frequently muzzled. Main roads were gradually cleared of poison to a distance of a chain (20 m) on each side (Erickson 1969, p. 60). When moving stock into unfamiliar areas, it became usual to send out scouting parties to identify and mark poison outcrops (P.G., 8 October 1842). On established properties, aborigines were temporarily employed in grubbing out poison plants in exchange for a ration of tobacco and flour.

The rate at which toxic species in areas settled by 1850 were identified, summarized in Table 3, is particularly revealing. The obvious conclusion to be made is that the commonest and most toxic species were among the first to be identified. River poison (*G. forrestii*), the major exception, has a restricted location on the rivers of the south coast. Of these, only the Kent and Hay Rivers were stocked before 1850 and then by small flocks grazed mainly on the upper reaches. Similar conclusions may be made for Stirling Range (*G. velutinum*), hook-point (*G. hamulosum*) and berry (*G. parvifolium*) poisons, all of which grow in rugged, hilly country or poorly grassed sandplains. The late discovery of the low toxicity poisons is almost self explanatory, but, in addition, crinkle leaf (*G. villosum*) and runner (*G. ovalifolium*) poisons grow along

Table 3

Toxic Species of Oxylobium *and* Gastrolobium *present in areas occupied by* 1850

Species	Toxic category	Maximum (a) toxic reading (ppm)	Date toxic effect determined	Date of first detailed description (pre 1864)
Box poison (*O. parviflorum*)	High level of toxicity (more than 1 000 ppm)	2 500	1842	1841
Heart-Leaf poison (*G. bilobum*)		2 650	1841	1829
Rock poison (*G. callistachys*)		1 000	1842	1844
Cluster poison (*G. bennettsianum*)		1 350	1841(b)	....
River poison (*G. Forrestii*)		1 200	1926	....
Champion Bay poison (*G. oxylobioides*)	Medium level of toxicity (more than 100 ppm)	1 050	1841(b)	1841
Sandplain poison (*G. microcarpum*)		600	1841(b)	1839
York Road poison (*G. calycinum*)		400	1841(b)	1841
Prickly poison (*G. spinosum*)		400	1901	1841
Berry poison (*G. parvifolium*)		300	1910	1841
Gilbernine poison (*G. rotundifolium*		150	1841(b)	1843
Hook-Point poison (*G. hamulosum*)		100	1920	1843
Stirling Range poison (*G. velutinum*)		300	1921	1853
Woolly poison (*G. tomentosum*)		n.a.	1955	....
Crinkle-Leaf poison (*G. villosum*)	Low toxicity (less than 30 ppm)	n.a.	1900	1841
Runner poison (*G. ovalifolium*)		n.a.	1910	1841
Bullock poison (*G. trilobum*)		n.a.	1921	1841

(a) Parts per million of the toxic radical in terms of air dried plant material.
(b) No real distinction was made between Champion Bay, York Road, Sandplain, Cluster and Gilbernine poisons because of their basic similarity. The toxic effect of all was effectively demonstrated by Drummond's experiments with Champion Bay poison (*G. oxylobioides*) in May 1841.

Source: Bentham 1864, v.2, p. 14–26 and 96–107; Gardner and Bennetts 1956, p. 40–77; Aplin 1967–71; Erickson 1969, p. 61–122.

the ground. Both were suspected in the early 1840's (Landor 1847, p. 380), but, perhaps because of their prostrate growth form, these suspicions were rejected. The thorny, unpalatable hardness of the leaves of both prickly and bullock poison (*G. trilobum*) explain their late identification as poisonous plants.

The second and more important conclusion is that there was no general knowledge transfer from one harmful species to others within the genera. Otherwise, all presently known toxic plants would have been quickly classified for, with the exception of crinkle leaf and runner poisons, all toxic species are easily recognised by the similarity of their growth form, leaf arrangement, and the shape, colour and arrangement of the flowers. Certainly cluster poison, Champion Bay poison, sandplain poison and gilbernine poison were identified when encountered but their similarity with York Road poison is very close indeed, clear distinctions only being possible when the plants are in flower.

That transfer was minimal indicates that no general framework for identification had yet been determined. To this extent, learning was still at a trial-and-error stage. It is possible, however, that this is indicative of an unwillingness to extend the range of harmful plants, and reflects the attitudes earlier adopted by Harris and others.

Learning characteristics

Three features of the learning sequence above are of particular significance in understanding the colonization of unfamiliar environments, namely: the role of problem solvers in a pioneering community; the relationship between a problem's difficulty and the rate at which it is resolved; and the characteristics of problem solving in pioneering situations..

The role of problem solvers

Although Curti (1957, p. 417-440) has demonstrated that individuals rather than formalized institutions are the key problem solvers in pioneering communities, neither Drummond nor Harris initially knew more about the poison problem than other colonists. They developed their competence through community pressure. Three factors account for their eventual dominance: they had superior knowledge in appropriate areas; they were able to isolate possible causes and solutions, and, as fellow pastoralists, they were viewed as status equivalents. They had credibility because they also suffered severe stock losses and were able to transmit their conclusions through informal channels. The deference of pastoralists was not complete, however, for they clearly reserved the right to question conclusions when these differed from their own views. Drummond and Harris were used to isolate causes and solutions but pastoralists were the final arbiters. This process minimized their own efforts while maximizing Drummond's and Harris' expertise and, as such, is an important modification of Found's observation that 'man attempts to optimize some utility while minimizing his own effort' (Found 1971, p.129).

The time factor in problem solving

The learning sequence indicates that the tempo of effective learning is not continuous but does accelerate up to the point where a solution is determined. This is a basic characteristic of all problem solving for much initial learning centres on definition and this is often surrounded by considerable confusion. Following definition, problem solvers are able to formulate and test hypotheses and are in a position to reject unsatisfactory solutions or reinforce those which seem suitable. Although new hypotheses may be added, there is a progressive decrease of possible alternatives. With resolution, there is a rapid diversification leading to both the solution of attendant problems and the development of compatible operational procedures. It must be emphasised, however, that problems can be effectively avoided before solutions are defined. This must seriously complicate attempts to identify adaptive processes for what may often be considered problem solving is, in reality, avoidance. That both are in response to stress levels adds a further dimension.

Colonizer problem solving

Problem solving is far from simple. Gagné (1970, p.36-69, 214-236) sees it as the apex of an eight-layered hierarchy which ranges from signal learning (essentially Pavlovian conditioning) at the base through stimulus-response connections where the learner acquires a precise response to one or more discriminated stimuli to concept development (common response to a general class of stimuli) and rule or principle learning where causal links between stimuli are clearly established. Problem solving requires competence in most of these learning types.

While the Gagne model identifies the stages of most learning situations, not all levels are applicable to the poison problem. The key problem solvers already had well developed learning patterns including single and multiple discriminations, chaining and associations, and, in the case of Harris, clearly established rules regarding the relationship between vegetation and stock deaths. What was critical was that these lower orders of the learning hierarchy had been developed in another and quite dissimilar environment and were thus inadequate. Because of this inadequacy, they had to be transformed or, more correctly, extinguished and replaced until they were compatible with the new environment. This was a major inhibitor of the learning rate.

The process of transformation may best be discussed by reference to the Lewinian concept of 'life space' (Lewin 1946, p.239-40). Lewin views behaviour as a function of the life space which is the product of a person's interaction with his environment and is essentially the known physical and psychological environment of that person as derived by his whole pattern of behaviour in a specific environmental setting. As the life space is highly structured, behavioural directions are well defined. When relocation occurs, a new and unstructured life space is imposed because there has been a complete change in the environmental component. The

individual is initially confused because of the lack of definite orientations but he institutes a learning sequence whose purpose is the *differentiation* of unstructured areas in the new life space (Lewin, 1942).

A similar viewpoint is expressed by Gerritz (1969) in his discussion of the effects arising from gross shifts in the maintaining environment. When the maintaining environment (analogous to Lewin's life space) is changed, the individual 'will bring to a new environmental setting . . . behavioural systems that have been maintained (and possibly acquired on the basis of) the stimuli in the setting from which he had come. It follows . . . that initial behaviour in response to the stimuli in the new setting will be a function of the similarity of those stimuli to the stimuli that controlled behaviour in the earlier context' (Gerritz 1969, p.119). If the new context is markedly dissimilar, severe constraints will be imposed on behaviour until the behaviour that does take place provides 'the basis for a new adaptive learning in connection with the stimuli available in the new setting'.

Both Lewin and Gerritz hold the view that relocation induces confusion and inhibits responsiveness to environmental stimuli. Yet, it is quite apparent that initial responses in Western Australia were quite pronounced and were accompanied by a degree of confidence that later responses did not have. Reference to Harris' assertion that stock died from 'blood striking' effectively demonstrates this point. That this was erroneous is irrelevant as it was not seen to be so. This example suggests that, in pioneering situations at least, differentiation is preceded by another process which can be termed *generalization*.

Generalization may be viewed as the process of interpreting and reacting to newly encountered environmental stimuli from the standpoint of already learned behaviour. Its dominant characteristic is the blanket application of well defined rules formed from past experiences as these are expressed through preconceptions. As such, it is essentially a function of factors internal to the individual. As well as established knowledge and preconceptions, these factors will include motives, goals and expectations. There is little observable interaction with (cognitive responsiveness to) external stimuli. It is only when blanket generalizations prove unsatisfactory that the search for the appropriate relationships between stimuli (differentiation) begins.

As suggested by the Gerritz quote above, differentiation refers to the process where specific relationships between environmental stimuli, both internal and external to the individual, are determined. It must be seen as a function of actions centred on the resolution of specific problems or the definition of particular behavioural orientations, and clearly involves an internal (nature of the problem solver) external (nature of the problem) dichotomy. That is, differentiation requires interaction between the problem solver and those environmental stimuli relating to the problem, the amount of interaction and hence differentiation increasing as learning progresses towards the point of ultimate resolution. This cannot be viewed as a purely perceptual or even more broadly cognitive process for affective components are also influential. As evidenced by Drummond's attitudes towards legumes, these are frequently inhibiting. Confidence levels may also inhibit (Found 1971, p.134). It is also apparent that when stress reaches a critical threshold all differentiation ceases and behaviour is characterized by avoidance.

From the preceding discussion, it is reasonable to postulate that all learning in colonizing situations progresses from the point where blanket generalizations are made to increasingly selective differentiation, both processes being embedded in a cognitive-emotional-motivational matrix. That no true separation of these components is possible is the problem facing all analyses of adaptive learning.

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